A CHRONOMETRIC ANALYSIS OF

THE EFFECTS OF ETHANOL

A thesis submitted in partial fulfilment

of the requirements for the Degree

of

Master of Science in Psychology

in the

University of Canterbury

by

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University of Canterbury

1983

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For Carolyn
Happiness and Peace

ACKNOWLEDGEMENTS

I would like to thank Paul Russell for his supervision and assistance,
Mr A. Patterson for the computer programming,
execQtype, Rotorua, for the typing and graphics,
staff of the Psychology Department at the University of Canterbury and my peers for numerous comments and suggestions,
those who volunteered and served as subjects,
my counselling colleagues for friendship and support,
"the Boys" for ensuring that humour prevailed when it was most needed – always!,
my mother for endurance and understanding,
and last but never least, thanks Foong May for being there.

ABSTRACT

Posner's (1978) simultaneous letter match task in which the interval between a warning signal and a letter was varied was used to examine the effects of ethanol on alertness. Control and experimental subjects, 10 per group, were given 0.1 ml alcohol per kg body weight and 0.69 ml alcohol per kg body weight to place their blood alcohol levels at about 5 mg alcohol per 100 ml blood and 80 mg alcohol per 100 ml blood respectively. Within each group, times to report the physical identity of letter pairs were faster than for pairs sharing the same name. Control subjects generally responded more quickly. For methodological reasons the effects of ethanol on alertness were not made clear by this experiment. Implications for future research are discussed.

CHAPTER ONE

INTRODUCTION AND LITERATURE REVIEW

1.1 INTRODUCTION

At a blood alcohol level (BAL) of 0.05 mg % (50 mg per 100 ml) typical behavioural effects are lowered alertness, impaired judgement, and release of inhibitions. At 0.10 mg % (100 mg per 100 ml) typical behavioural effects include slowed reaction times and impaired motor function (Ray, 1978, p.146).

Alertness is one of three basic components in the study of attention and can be defined as an ability to develop and maintain an optimal sensitivity to external stimulation (Posner 1975, 1978; Posner & Boies, 1971). To attend efficiently to, especially, external events, subjects must be in a general state of alertness. The effects of this state seem best to be described as reducing the time for some central mechanism to respond to the buildup of information about a signal (Posner, 1978).

It is on this anecdotal and theoretical background that this thesis is based.

Although a behavioural effect—lowered alertness—is reported at an approximate blood alcohol level of 50 mg per 100 ml; no definition or supporting research is quoted by Ray (1978).

With the observation by Fleishman and Bartlett (1969) that there is still a need for studies which evaluate the effects of specific drugs and dosages on specific classes of performance, also in mind, the current experiment examines the effects of ethanol on alertness using a paradigm devised and used by Posner and colleagues (Posner & Boies, 1971; Posner, Boies, Eichelman & Taylor, 1969; Posner, Klein, Summer & Buggie, 1973; Posner & Mitchell, 1967; Posner, Nissen & Klein, 1976).

1.2 LITERATURE REVIEW

This review comprises three sections. The first section is concerned with the extent of alcohol-cognitive research and what is known about the effects of alcohol on the central nervous system (CNS) and behaviour. The second area reviewed covers the theoretical and experimental development of the concept of alertness. Finally, the third section integrates the information from the previous two sections and presents it as a set of predictions of hypotheses which the current experiment is designed to test.

The focus of the investigation is with ethyl alcohol or ethanol (C_2H_5OH), and alcohol refers specifically to ethanol. The two terms will be used interchangeably.

1.2.1 Alcohol-Cognitive Research

Alcohol and its effect on cognitive processes is a widely researched area. This research encompasses topics such as temporal information processing (Goldstone et al, 1977, 1978), the rate at which information can be processed (Moskowitz & Burns, 1971; Moskowitz & De Pry, 1968; Moskowitz & Roth, 1971), memory (Birnbaum & Parker, 1977; Birnbaum et al, 1978; Jones, 1973; Jones & Jones, 1977), general cognitive performance (Jones & Vega, 1972, 1973; Lubin, 1977) and the processing stage affected by alcohol using Smith's (1968) four stage information processing model (Huntley, 1972, 1974, Tharp et al, 1974). With respect to reaction time data, alcohol lengthens the time to respond to the target stimulus and increases the variability of that responding (Teichner, 1954; Cass & Frederick, 1961).

Thus we have a lot of information about the action of alcohol and although there is some understanding of its physiological and behavioural effects (Wallgren & Barry, 1970; Ray, 1978), specific knowledge as to mechanisms of action and processes affected, as Dietrich (1975, cited in Ray, 1978) and Fleishman and Bartlett (1969) have commented, is lacking.

Evidence suggests that alcohol affects our ability to process information properly. Perception of the situation or capability of responding is not impaired, rather the ability to select and organise the correct response is affected (Huntley, 1972, 1974; Tharp et al, 1974).

Any improvement in performance following alcohol consumption is largely attributed to disinhibition of areas of the brain (Ritchie, 1975, cited in Ray, 1978). However, Murphree (1973) has suggested, from EEG recordings, that in some individuals alcohol may induce alerting and activation. This is largely attributed to catecholamine release.

The effect of lowered alertness at 50 mg per 100 ml blood, as cited by Ray (1978), may not hold true. Instead, there could be initial increase in alertness and performance due to disinhibition and catecholamine release before the more slowly developing and greater depressant effects of alcohol become predominant with resultant decreases in alertness and performance.

Research involving alcohol needs to concern itself with two issues that arise as a consequence of its use.

First, the effect of alcohol on the central nervous system is directly proportional to the level of alcohol in the blood, and the rate at which the blood alcohol level rises is a factor in determining behavioural effects (Jones & Vega, 1972, 1973; Ray, 1978).

Second, behavioural and CNS tolerance to alcohol develops with prolonged use which can affect subject selection and subsequent statements about the effects of certain dosages on performance.

As a result of tolerance and potential subject reactivity to alcohol, any dose-response curve contains considerable variability and at best, only general behaviour can be reported (Ray, 1978, table 7-7, p.146).

Thus we are left with a broad picture of the effects of alcohol on behaviour and the central nervous system, and with the possibility that the effect of reduced alertness at about 50 mg alcohol per 100 ml blood, reported in the introduction, in some doubt.

1.2.2 The Concept of Alertness

Over the past 70 years, alertness has been studied under the effects of certain drugs, notably alcohol (Murphree, 1973), caffeine (Regina et al, 1974), and chlorpromazine (Tecce et al, 1975), but more often as a component of attention.

Woodrow (1914) noted that with constant block foreperiods or preparatory intervals, maximal adaptation of attention occurs at about 2 seconds. Teichner (1954) found that a preparatory or warning signal yielded faster reaction times than did omission of such a signal. Readiness to respond or set depended on the length of time between the warning signal and the target stimulus and was in the range of 1.5 to 8 seconds. Klemmer (1956) observed, however, that with minimum variability in the foreperiod, and hence reduced subject uncertainty about the time of occurrence of the stimulus, the optimum mean foreperiod was less than one second.

Research by Bertelson and his colleagues in the 1960's further contributed to our understanding of alertness or preparation.

Bertelson and Boons (1960) concluded that preparation is not a completely selective process, a statement which was to be reiterated by Thomas (1974) and Holender and Bertelson (1975).

Bertelson (1967) undertook the first major study of the time course preparation. An auditory warning signal could be used as a time cue to start preparatory adjustments without initiating a refractory period (a period following a stimulus in which responding is delayed). It also caused an immediate facilitation of the reaction time to the visual target stimulus.

Bertelson (1967) had shown that preparation can be built up much faster than the 2 to 4 seconds suggested by Woodrow (1914).

In 1968, Bertelson and Tisseyre suggested that preparation was not a process which engaged central decision mechanisms in an intermittent way. That is, once preparation is effected, the analysis of the target stimulus need not be delayed until it is completed; the shift from preparation to reaction can occur at any time.

Finally, Bertelson and Tisseyre (1969) replicated Bertelson's 1967 study, this time employing a visual warning signal. Similar results were obtained and this was taken as further evidence that the warning signal does not start a refractory period but gradually installs a state of heightened responsiveness or preparation.

Although he had made a major contribution to the field, Bertelson (1967) felt that a comprehensive picture of the phenomenon was lacking because there had been no systematic study of the effects of the duration of constant foreperiods below one second. His own experimentation was leading to such a position, and in the same year, a study entitled "The Chronometric Analysis of Classification" (Posner & Mitchell, 1967), introduced an experimental paradigm that was to make such an analysis possible and ultimately, was to refine our understanding of the concept of alertness.

The paradigm is as follows: after a warning signal (e.g. a cross), a stimulus pair—letters, digits, or forms—is displayed which requires a same-different response (i.e. "same" if the elements of a pair are identical, and "different" otherwise), indicated by pressing one of two keys. The response time is recorded, feedback given, and following a variable interval, the next trial is initiated.

In these experiments, identity (for a "same" response) can be defined in a number of ways. For example, in a physical identity match (PI) task subjects are instructed to respond "same" only when stimuli are physically identical (e.g. AA). A name identity match (NI) task requires subjects to respond "same" whenever a pair of stimuli share the same name (e.g. Aa). Physical matches are made about 40 msec faster than name matches. Even in a name match task, physically identical stimuli are matched faster. That is, where a subject is instructed to match on the basis of common names, the likes of AA are responded to more rapidly than the likes of Aa by about 70 msec.

Posner (1978) has argued that two distinct processes are involved. In order to make a name match for physically non identical stimuli (e.g. Aa), some long term memory name code has to be accessed. By contrast, physical identity can be established without recourse to any stored knowledge.

Thus, the difference in response time for NI and PI tasks has been interpreted as indicating the time required to look up or access records in long term memory. This difference has been exploited in the study of development (Kraut, 1976), ageing (Bisanz et al, 1979), and intelligence (Hunt, 1980).

Evidence for a generalised alerting component derives from the fact that both same and different responses vary in the same way with the interval separating a warning signal

from the stimulus pair. Response time decreases quite sharply as the warning interval (WI) increases from 0 to 500 msec. Thereafter RT slowly increases. This pattern is true of name match and physical match conditions.

These results suggest that it takes about 500 msec to achieve a state of alertness, and hence optimal performance. The process of achieving this level of alertness is referred to as preparation. In addition, Posner et al (1973) concluded that alertness does not affect the build-up of information within the memory system but only the rate at which a later system responds to the information. Increased alertness produces a reduction in RT but no reduction in the numbers of errors. In fact, errors increase. There is a speed-accuracy trade off.

Posner's 1975 article further improved our theoretical understanding of the concept.

Seen within an information processing framework, alertness referred to a state of the organism which affects general receptivity to incoming information and in particular, interrogation and response to that information.

Alertness was seen as having tonic and phasic aspects, the latter having been the focus of research outlined above, and referred to the changes in alertness occurring in the period between a warning signal and a target stimulus. Tonic aspects referred to changes in alertness occurring during the course of the day or throughout the life cycle.

Physiological evidence (Sharpless & Jasper, 1956; Webb & Obrist, 1970; Gazzaniga & Hillyard, 1973) was seen as supporting such a dichotomy and suggestive of the location for the control of such changes in alertness.

The 1978 work, Chronometric Explorations of Mind, summarised Posner's experimental and theoretical position based on 10 years or so of research. Included in this summary was a concise description of the concept of alertness as Posner understood it. Bertelson (1967) had been answered.

1.2.3 Implications for Alcohol Research

We can summarise the major points from the above two areas and from this, predict what results and effects we should obtain using the paradigm previously outlined and under the influence of alcohol.

At about 50 mg alcohol per 100 ml blood there is a decrease in alertness (Ray, 1978) although this view has been challenged (Murphree, 1973). Alcohol tends to lengthen reaction time and increase the variability of responding (Teichner, 1954; Cass & Frederick, 1961). Our ability to process information properly is also affected (Huntley, 1972, 1974; Tharp et al, 1974).

From Posner's work we know that we are in a state of maximal alertness to respond with a warning interval of about 500 msec (Posner & Boies, 1971); that phasic alertness varies rapidly over short periods of time; and that we respond faster to stimuli that are physically identical than to stimuli with the same name.

The present study employed two groups. A control group with blood alcohol levels (BALs) estimated to be near 5 mg alcohol per 100 ml blood, and an experimental group with BALs near 80 mg per 100 ml.

We would expect that the basic findings of greater response time to stimuli sharing the same name (e.g. Ff) than to those which are physically identical (e.g. AA), and for response times to decrease and for errors to increase, with increases in warning intervals in the range of 0 to 1000 msec, to be true of both groups. However, we would expect the RTs of the experimental group to be longer in all stimulus and warning interval conditions, and for the experimental group to reach maximal alertness (i.e. minimum RT and maximum errors) at a longer warning interval.

That is, we would expect a replication of the Posner and Boies (1971) results in subjects with significant and positive BALs.

We would predict that, in general, the RTs of those in the experimental group would be elevated, not as a result of motor impairment but because the necessary cognitive components take longer to respond—a consequence of a decrease in alertness. Subsequently, we would expect maximal alertness to be reached at increasingly longer warning intervals with increased BAL.

If, however, performance by the experimental group is superior to that of the control group, this would tend to support Murphree's (1973) view of an initial increase in alertness which heightens performance, before the more depressant action of alcohol predominates which results in a decrease in performance (i.e. increased RT).

We are now in a position to put these predictions to the test using the Posner paradigm. The next chapter details how this was attempted.

CHAPTER TWO

METHOD

SUBJECTS

From a pool of volunteers, twenty people were selected to serve as subjects in the experiment. They were assigned at random, 5 male and 5 female, to a placebo control (estimated BAL, 5 mg/100 ml), and an experimental (estimated BAL, 80 mg/100 ml) group. They were aged between 17 and 28.

The subjects were selected on the basis of the self reported drinking pattern as determined by questionnaire. This questionnaire was abstracted from Bowman et al (1975), Elvy (personal communication), and Prebble (1980). Subjects were selected if they were within the criteria for being classified as a moderate or social drinker, as determined by the author, using guidelines abstracted from Bowman et al (1975). Analysis of drinking patterns was based on both quantity and frequency of alcohol consumption.

Consumption ranged from subject 9, who reportedly drank an average of 1.00 ml of alcohol per day, to subject 16, who drank an average of 43.33 ml per day.

The median consumption of the placebo group was 5.41 ml per day, while that for the experimental group was 5.42 ml per day.

The median was chosen because it indicates the amount of alcohol consumed by the person who stands at the midpoint of the consumption distribution of all drinkers (Casswell, 1980).

The questionnaire and drinking patterns of all subjects appear in Appendix 1.

All subjects were paid $5 an hour for their participation.

APPARATUS AND STIMULI

Experiments were run on an Apple II Plus computer with displays presented in black and white on a 22 inch (560 mm) Phillips KTV.

The computer generated the letter pair displays from a 5-letter alphabet. With these letters, each upper and lower case, a total 100 letter pair arrangements is possible. These 100 pairs consisted of:

(a) 10 physical identity (PI) letter pairs, e.g. AA, ff;
(b) 10 name identity (NI) letter pairs, e.g. Bb, Hh;

making 20 'Same' items and;

(c) 80 'Different' (D) letter pairs, e.g. hA, FB.

Items were randomly selected for display with the only restriction being that each block of 40 trials contain 20 'Same' items—10 PI and 10 NI.

The stimulus alphabet was constructed in the computer's High Resolution Graphics from shape tables derived from Letraset Data 70. The letters are presented in their Letraset form in Appendix II.

Letter pairs were displayed in a 40 x 20 mm area, commencing 180 mm from the left and 120 mm from the top of the screen. On average, the lower case letters were 14 mm wide with a 10 mm gap between the pair, and the upper case letters were 18 mm wide with a 4 mm gap between the pair.

A trial consisted of a warning signal (WS), a warning interval (WI), a letter pair requiring a response, performance feedback, and a long (8—15 sec), randomly determined inter-trial interval.

The WS was a cross, 26 mm long, 22 mm wide and with an 8 mm stroke width. Its leading edges were 180 mm from the left and 90 mm from the top of the screen. It was displayed for the length of the WI. All displays subtended no more than 1.5° of visual angle at a viewing distance of 1.6 metres.

Warning intervals of 50, 100, 200, 500 and 1000 msecs were examined. Each WI trial block consisted of 10 practice trials, which were not recorded, followed immediately by 40 experimental trials. Following Posner and Boies (1971), the WI was constant for a block of trials, but varied between blocks. Order of block presentation was determined by a latin square.

Following the cross and directly below it, a pair of adjacent letters was displayed to which the subject responded "same" if they had the same name or "different". A voice

activated relay was used. The display remained visible until the relay was activated.

Performance feedback gave information to the subject about the speed of response—fast, average or slow—relative to the running mean, and whether the response was correct (display "correct") or in error (display "wrong").

Figure 1, modified from Hunt (1980, p.451) explains the paradigm used in the current investigation.

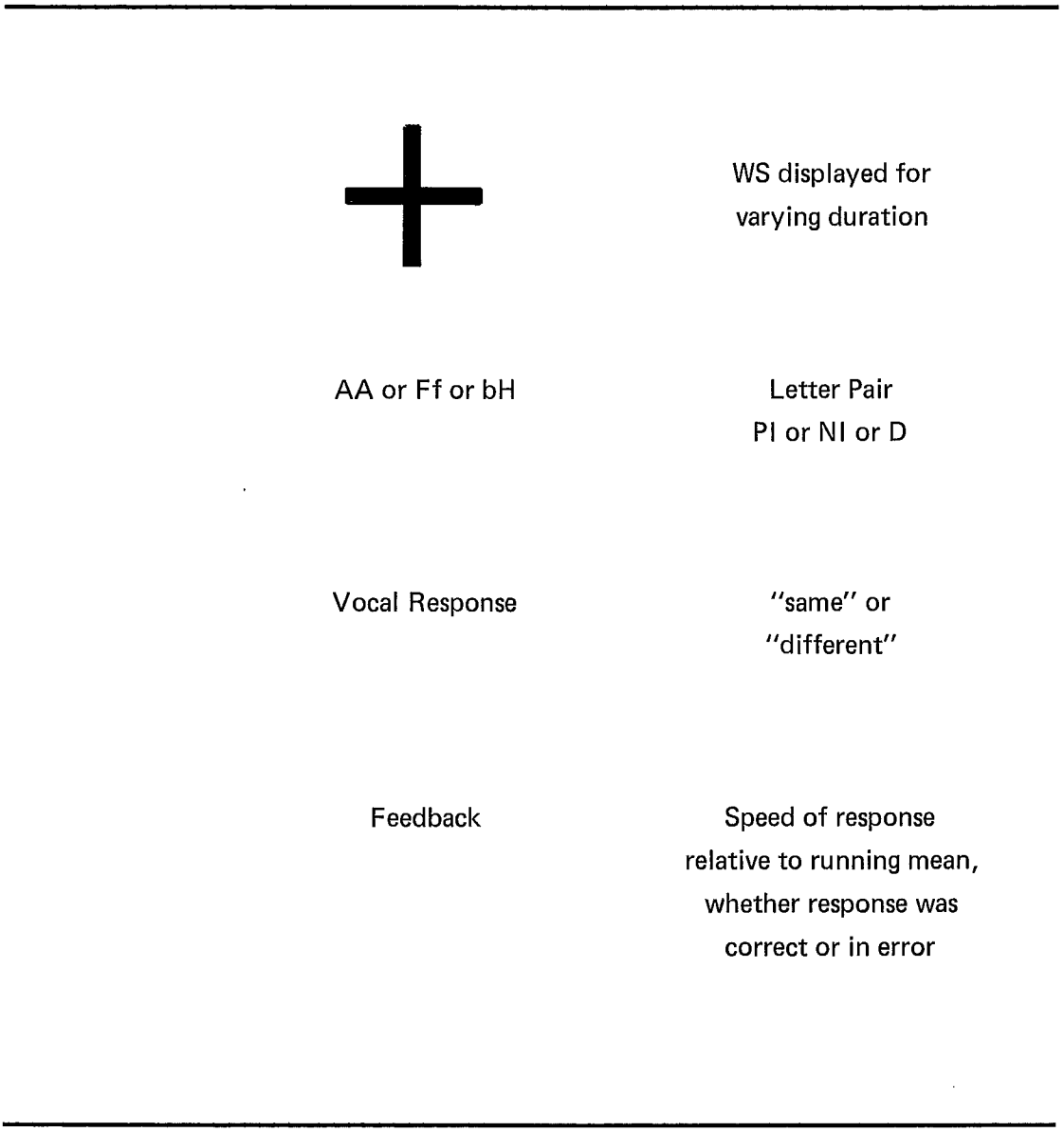

FIGURE 1: Stimulus Identification Paradigm

If the subject made an error, the trial was discarded and not recorded. Two other trials were inserted later in the block. One of the extra trials matched the error in stimulus type, i.e. PI, NI, or D. Only the extra trial identical in type to that to which an error was made was recorded and included in the analysis. Re-presenting both a 'Same' and a 'Different' stimulus preserved the 50/50 balance of "same" and "different" responses.

Whenever the subject responded "same", the experimenter depressed a button until a tone sounded in an earphone. This enabled the computer to identify erroneous responses.

DOSE

The experimental treatment dose was 0.69 ml of alcohol per kilogram of body weight, and was made up to 300 ml with orange juice. Vodka, 43% ethanol by volume, was the beverage used. This dose was designed to put the blood alcohol level (BAL) at about 80 mg per 100 ml one hour after drinking.

The placebo control dose was 0.10 ml of alcohol per kilogram of body weight, made up to 300 ml with orange juice. This gave an approximate BAL of 5 mg per 100 ml one hour after drinking.

PROCEDURE

Subjects were randomly assigned to receive the experimental or placebo dose— 5 males and 5 females per condition. All subjects knew that the experiment involved the consumption of alcohol, but they were not told the dosage received or what treatment condition they were in until the task was completed.

Prior to the experimental session, subjects attended a meeting where measurements of height and weight were taken. This was done for the purposes of dosage calculation and estimation of BAL .

No subject reported being on medication. They were asked this for their own welfare and to avoid possible confounding of any treatment affects.

Subjects were requested not to consume any drugs on the day of the experiment and to fast in the four hours preceding the start of the experimental session. A verbal check verified compliance with this request.

Upon arrival for the experiment, the task was outlined to the subject.

They were given 15 minutes to consume the appropriate beverage, and all did so with ease. For convenience and ease of consumption, the drink was divided into two equal portions of 150 ml.

Following consumption of the drink there was a 25 minute absorption period. During this period, the task was explained in more detail and one block of trials given to facilitate familiarity with the procedure. Experimental trials—in five, 40 trial blocks,—now commenced, and took approximately 80 minutes to complete.

BAL was estimated at 40 minutes and 120 minutes after drinking began, using formulas derived by Watson and Batt (1976) and Watson, Watson, and Batt (1980). These appear in Appendix III. The mean estimated BAL for both dose conditions is shown in Table 1.

TABLE 1

Mean Estimated BAL (mg per 100 ml)

Condition	Minutes After Drinking	
	40	120
Placebo	6	0
Experimental	88	71

During the task, subjects were seated in a comfortable position in front of the display unit. The subject was told to respond "same" or "different" on the basis of the name identity of the letter pairs and to aim for maximum speed and minimum errors.

The experimenter was present throughout the session, monitoring responses and recording computer output. This enabled the subject to have a break between a block of trials.

All testing was done between the hours of 2pm and 4pm or 5pm and 7pm.

A relaxed friendly atmosphere prevailed and at the end of the session, sandwiches and fruit was made available to all subjects.

CHAPTER THREE

RESULTS

The effects of the variables Dose, Warning Interval (WI) and Stimulus Type (PI, NI) on reaction time (RT) were examined in a 3-way Analysis of Variance with repeated measures on WI and Stimulus Type using the BMD O8V programme.

At each WI, subjects made 40 responses – 10 PI, 10 NI and 20 D. The median RT for each stimulus type at each WI formed the raw data for analysis. That is, 15 measures per subject. Median RTs were used because they are less affected by outlying observations.

Because results from the 'Different' condition relate to none of the predictions made in the present study, and are of no theoretical importance for the study no analysis of them is presented.

A summary of the results for the two groups for each Stimulus Type at the various WI appears in Table 2.

(See page 15).

The relatively low percentage of errors is a feature of subject's responding and is consistent with past research.

Now examining the hypotheses from Chapter One in turn.

It was posited that RTs of those in the experimental group would be longer than RTs of subjects in the placebo control group. That is, there would be a Dose main effect – the mean RT of the experimental group, averaged over all stimulus and warning interval conditions, will exceed that of the placebo control group. However, the results do not confirm this prediction. The Dose main effect is not significant, $F(1,18) = 0.02$, $p > 0.05$, and no interaction involving Dose was significant.

A second set of predictions related to Posner's past research and it was expected that those basic findings would prevail in the current experiment. They were:

Firstly, that RT will vary as a function of WI for all stimuli (PI, NI), with RTs decreasing in the range 0–150 msec and thereafter remaining constant or increasing. The relevant data is displayed in figure 2.

(See page 16).

Examination of the figure indicates that the data approximately follows this pattern. However, the WI main effect is non-significant, $F(4,72) = 1.51$, $p > 0.05$. The WI x Stimulus Type interaction was also not significant, $F(4,72) = 2.59$, $p > 0.05$. Further, Posner (1978) argues that as a subject becomes alerted, response time falls and errors increase. The lack of any WI effect in the present data makes it impossible to establish any such speed-accuracy relationship.

Secondly, it was predicted that if alcohol retards the level or rate of onset of alertness then the response curve for the experimental group would be displaced towards the right of that for the placebo control group or be considerably flatter. That is, a significant Dose x WI interaction is predicted. The effect was not significant, $F(4,72) = 1.09$, $p \not< 0.05$.

Thirdly, RT to PI stimuli will be faster than RT to NI stimuli and by the same amount at each WI. That is, we expect a Stimulus Type main effect but no Stimulus Type x WI interaction, and we further expect this to apply to each group. The Stimulus Type main effect was significant, $F(1,18) = 28.29$, $p < 0.001$. The Stimulus Type x WI interaction was not significant, $F(4,72) = 2.59$, $p \geq 0.05$.

It is possible, from Hunt (1980), that the NI–PI difference will be greater for the experimental group, and a significant Dose x WI x Stimulus Type interaction is predicted. The relevant data is graphed in Figure 3.

(See page 17).

The effect was not significant, $F(4,72) = 1.83$, $p > 0.05$.

TABLE 2

Means, Standard Deviations (in msec) and Percentage Error of Placebo Control and Experimental Groups

Group		Warning Interval (msec)				
		50	100	200	500	1000
Placebo Control	PI Mean	537	528	548	544	580
	SD	86	83	119	102	121
	Error	0.69	0.23	0.95	0.24	0.23
	NI Mean	646	557	599	626	596
	SD	133	129	147	123	155
	Error	2.53	2.58	1.67	0.96	3.04
Experimental	PI Mean	559	583	513	563	557
	SD	109	146	80	110	92
	Error	0.89	0.47	0.91	1.14	0.00
	NI Mean	645	598	566	597	645
	SD	168	147	126	144	78
	Error	5.13	2.11	2.95	3.18	1.21

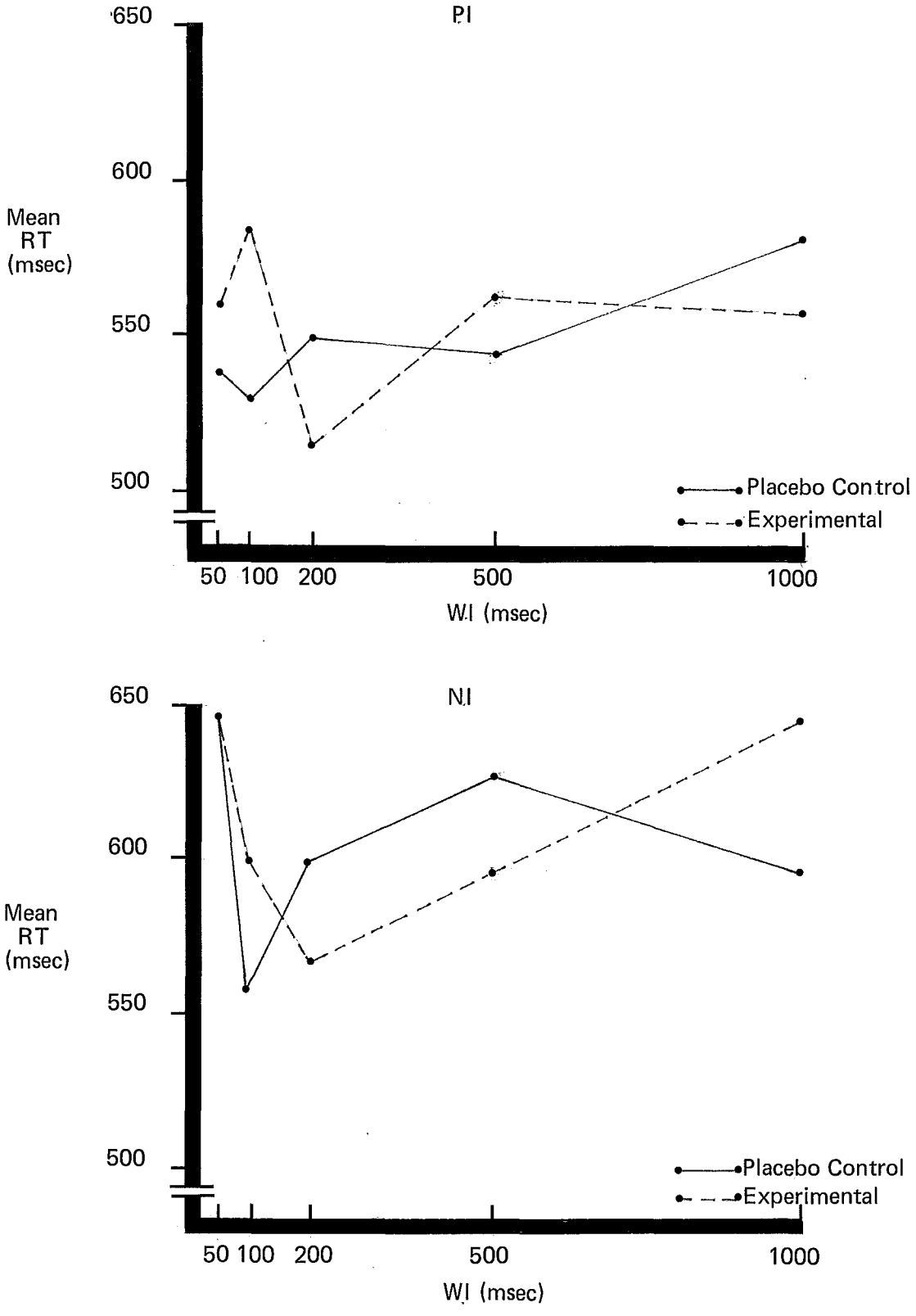

FIGURE 2: Mean RT (msec) to PI and NI Stimuli

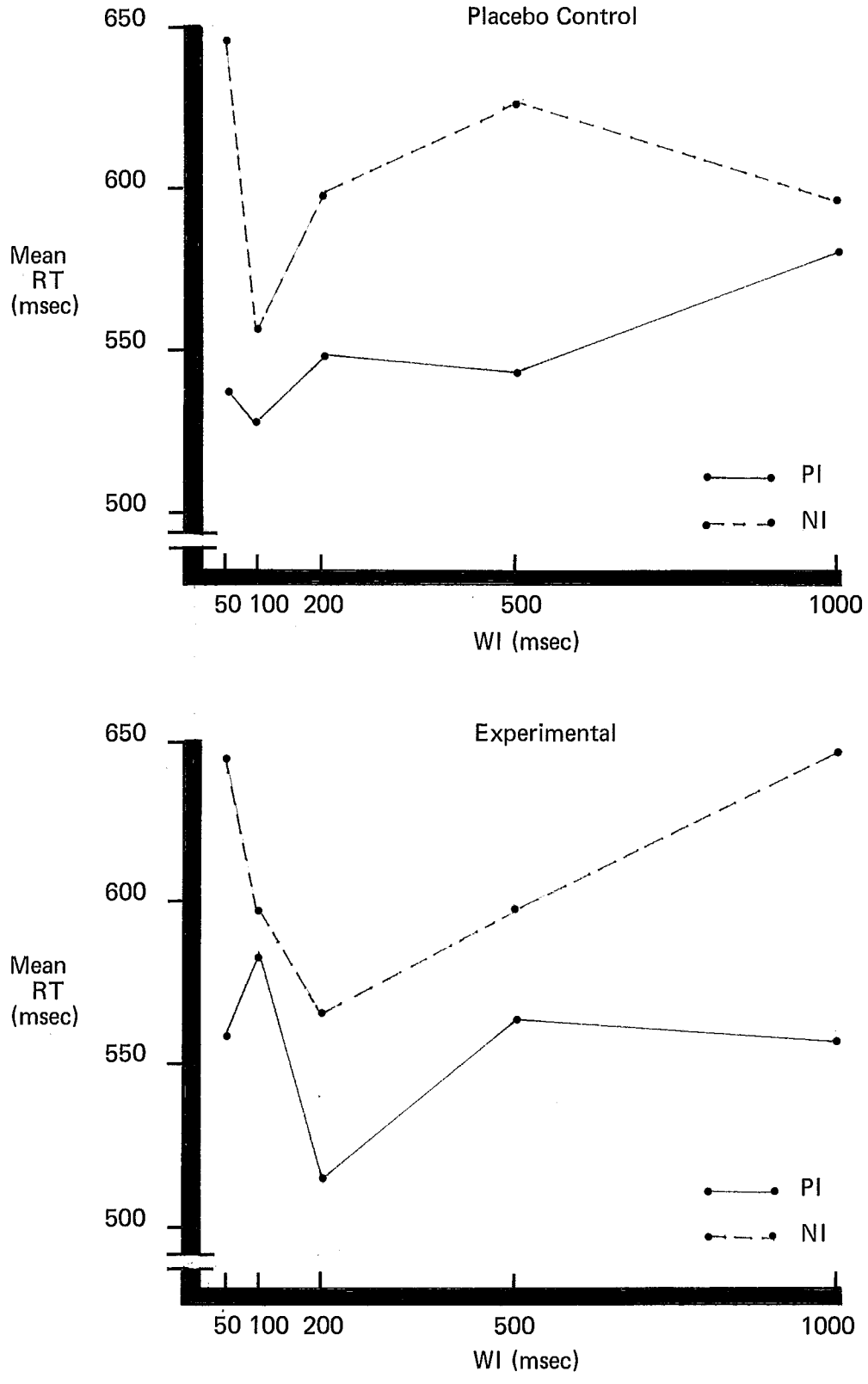

FIGURE 3: Mean RT (msec) by Groups to Stimuli

CHAPTER FOUR

DISCUSSION AND CONCLUSIONS

A major difficulty in interpreting the hypothesized effects of alcohol on alertness arises in the present experiment because the basic effects of alertness on RT and accuracy which have been reported by Posner and Boies, (1971) were not replicated on either the placebo control or the experimental groups. Three methodological differences between Posner's work and the present experiment may account for this.

Firstly, the mode for the response was not the same in the two tasks. Posner has consistently employed a key depression response in his research whereas this study employed a voice response. The responses obtained under each system may be qualitatively different. A vocal response may be more compatible with the stimulus type than an arbitrary left key for "same" and right key for "different" response.

Secondly, the stimuli used differed in both quality and quantity (a 15 letter alphabet in Posner and Boies, 1971, versus a 5 letter alphabet in this task). Quantity per se would not be expected to have much effect. Even if a smaller stimulus set, with its consequent greater number of presentations per stimulus, had led to an automized "same" or "different" response to each stimulus, we would, on the basis of Posner's work (Posner, 1978), still expect RTs to fall with warning intervals in the range 0 to 150 msec. However, the relatively novel Letraset Data 70 letters used (see Appendix Two) may have masked other effects. Several subjects reported that the letters were confusing.

Thirdly, and possibly most importantly, a number of factors contrived to allow practice effects to mask other experimental effects. Posner and Boies (1971) collected data on four separate days and treated the first day's results as practice, and reported no day effects on days 2–4. Because BALs cannot be engineered on several occasions nor sustained for prolonged periods, only a single session with limited trials was employed in the present experiment. Consequently RTs were probably falling rapidly, due to practice, as trials progressed. Thus, RTs to later blocks would be faster than those to earlier, for all warning intervals. This practice effect (perhaps even more pronounced because of the novel letters) together with the blocked presentation of trials at each WI (a procedure adopted from Posner and Boies, 1971) would, despite the fact that order of presentation of WI blocks followed a Latin Square, result in an insensitive design in which practice masked the effects of alertness.

The discussion now turns to the hypotheses in light of the obtained results.

It was predicted that RTs of the experimental group would be longer than those of the placebo control group. This did not occur. This could be due to the use of a vocal response

as mentioned above. While the literature provides numerous examples of slowed RTs with ethanol, this author knows of none using vocal response.

Secondly, it was predicted that if ethanol affected levels of alertness, then either RT would not fall with WI, the commencement of fall would be delayed, and/or the rate of fall would be less steep. The failure to find any affect of WI on the RTs of the placebo control group precludes any evaluation of this hypothesis.

Thirdly, as pointed out in the introduction, "same" responses to physically identical stimuli are invariably faster than to physically different stimuli sharing the same name. The NI–PI difference has been taken to indicate the speed of access of information in long term memory (Hunt, 1980). It was suggested that the speed of access to stored information may become slower as a consequence of ethanol. While the basic finding of slower "same" reponses to NI than to PI stimuli at all WI was true of the present experiment, the NI–PI difference was the same for both groups. It seems safe to conclude that at the BALs under consideration, alcohol does not affect the speed of access to information in long term memory.

A number of improvements in research design are needed if the effects of ethanol on alertness are to be assessed. For obvious reasons the number of occasions on which subjects ingest alcohol, and the duration for which BALs are elevated is limited, and as we have seen this results in difficulties in controlling practice effects. Following Posner, trials were blocked according to WI. An alternative which would better control practice effects within the present time constraints would vary WI at random from trial to trial. Another possibility is to run subjects on practice sessions perhaps over a number of days until asymptotic RTs are realized, before introducing the treatment of ethanol.

The particular letter set used resulted because at the time this was the only set available on the local Apple system. A more conventional alphabet is now available.

Third, it would be beneficial to use a blood test to get an accurate blood alcohol level, so that one is in a position of knowing at what level of intoxication the subject is performing under. This helps to satisfy Fleishman and Bartlett's (1969) criticism of the need to evaluate the effects of specific drug dosages on specific classes of performance.

Finally, there may be merit in incorporating the suggestions of Vogel–Sprott (1976) who advocated repeated testing at low alcohol doses to more accurately measure the effects of ethanol on performance under conditions approximately those in the "real world".

Perhaps it is best to view this research as going some way towards answering the comments of Dietrich (1975, cited in Ray, 1978), that the body of information regarding the action of ethanol in the brain far exceeds the body of knowledge; and Fleishman and Bartlett (1969), that there is still a need for studies which evaluate the effects of specific drugs and dosages on specific classes of performance.

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APPENDICES

Appendix One: Questionnaire and Drinking Patterns

Appendix Two: Stimulus Alphabet

Appendix Three: Formulas for Estimating BAL

Drinking Pattern Questionnaire

The purpose of this questionnaire is to establish the drinking patterns of people. This will be used to select subjects to participate in research examining the effects of alcohol on performance. All information will be treated as confidential. Please be as accurate as you can and answer every question.

SEX: Male _____ Female _____ AGE: _____years

(1) When did you last drink any alcohol?

_____ months ago
_____ weeks ago
_____ days ago
_____ hours ago

(2) On an average drinking occasion, how much alcohol would you consume? (example: 2 jugs beer or 3, 5oz glasses wine or 2, 12oz glasses beer)

Beer: _____
Spirits (specific beverage): _____
Table Wine: _____
Fortified Wine (sherry, port, vermouth): _____
Other (specify): _____

(3) On average, how long is it between drinking occasions? (circle one)

A less than one day
B two or four days
C five to thirteen days
D two or three weeks
E about a month
F more than one month

Thank you for your co-operation. In the event that you are selected to participate in research examining the effects of alcohol, would you provide the information asked for below.

NAME: _____
CONTACT: _____
SUITABLE TIMES FOR PARTICIPATION: _____

Drinking Patterns of all Subjects

Group	Subject	Consumption (ml alcohol per day)
Placebo Control	1	21.00
	2	6.67
	3	26.67
	4	16.67
	5	18.33
	6	2.67
	7	4.17
	8	2.92
	9	1.00
	10	3.53
	median	5.42
Experimental	11	5.00
	12	3.33
	13	4.39
	14	4.17
	15	40.00
	16	43.33
	17	6.67
	18	5.83
	19	2.61
	20	26.89
	median	5.41

Stimulus Alphabet

The above letters were used as the stimulus alphabet in this investigation, and are from Letraset Data 70.

Formulas For Estimating Bal

1. Total Body Water (TBW)

For Males:

TBW = 2.447 - 0.09516 A + 0.1074 H + 0.3362 W
(litres) (age in years) (height in cm) (weight in kg)

For Females:

TBW = -2.097 + 0.1069 H + 0.2466 W
(litres) (height in cm) (weight in kg)

2. Estimation of BAL

$$c_t = \left(A(g) - \frac{0.13t \times TBW}{0.8}\right) \times \frac{0.8}{TBW}$$

Where c_t is the BAL in grams per litre
A(g) is the alcohol dose in grams
t is the time in hours after drinking
and TBW is the total body water in litres.